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SHORT REPORT

# False Lumen Embolization for Type B Dissection Complicated by Hemoptysis

**R. López-Benítez,* G.M. Richter, A. Luburic, D. Böckler, G. Kauffmann and P.J. Hallscheidt**

*Department of Radiology, Heidelberg University Klinikum, Im Neuenheimer Feld 110, 69120 Heidelberg, Germany*

*In this report, we describe successful treatment of a patient with hemoptysis by false lumen embolization of a type B aortic dissection.*

*Keywords: Type B dissection; Embolization; Hemoptysis; Aortobronchial fistel.*

Association of aortic dissection and aorto bronchial fistula (ABF) has been reported in a few cases, especially in those patients who have a previous history of aortic repair.[1–5] Urgent treatment is imperative in order to avoid fatal massive hemoptysis.

Since, the bronchial circulation is the major source of hemoptysis, therapeutic embolization of bronchial arteries can be performed to stop the bleeding.[6]

In this report, we describe successful treatment of a patient with hemoptysis by false lumen embolization of a type B aortic dissection.

To our knowledge there are no current reports in medical literature of embolization of the aortic false lumen as a therapeutic option after thoracic aorta stent-graft implantation to close the source of bleeding in progressive hemoptysis.

## Case Report

A 78-year-old man presented in March 2003 with progressive and recurrent episodes of hemoptysis shortly after the implantation of two thoracic stent-graft prostheses for the treatment of a chronic type B dissection. The patient's medical history included hypertension, cutaneous T-cell lymphoma, and open repair of an asymptomatic infra renal aortic aneurysm in February 2001. The patient had a long history of smoking (45 pack-year). He denied other drug use. He had a regular diet, and the family history was unremarkable. A type B dissection has been diagnosed in September 2002 after a period of intensive thoracic pain.

The type B dissection was followed up from September 2002 until March 2003 by CT scans. No shrinking or false lumen thrombosis was observed. So on the 28th of March 2003 two thoracic stent-grafts were implanted at the thoracic level T5–T10 (Talent, Medtronic Tolochenaz, Switzerland). In August 2003 the patient was re-admitted with recurrent episodes of hemoptysis. Clinical examination revealed a thin adult, with the following signs: pale skin, temperature of 39.7 °C, slight tachypnea, tachycardia (120 bpm), blood pressure of 110/60 mmHg, and normal neurological status. Lung auscultation revealed the presence of minimal bilateral diffuse rales; however, the plain chest radiograph showed no significant changes, just a mild enlargement of the mediastinum.

Initial laboratory tests demonstrated a red blood cell count of 2.9 Pl, white blood cell 5.71/nl, hemoglobin 8.5 g/dl, platelets 393/nl, APPT 24.3, INR 1.1. Other blood chemistries were normal including

*Corresponding author. Dr Ruben López-Benítez, MD, Department of Radiology, Heidelberg University Klinikum, Im Neuenheimer Feld 110, 69120 Heidelberg, Germany.
*E-mail address:* rlbmex@hotmail.com

1078–5884/000370 + 06 $35.00/0 

electrolytes and serology for infectious diseases. Because of the febrile status, blood cultures were obtained and prophylactic wide-spectrum antibiotic therapy was started with amoxicillin and clavulanate (augmentan 850 mg. GlaxoSmithKline Munchen, Germany). Shortly after medical treatment, the fever resolved and laboratory inflammatory parameters normalized, however, the initial blood culture result 72 h after they were obtained revealed the presence of *Staphylococcus aureus*.

Thorax CT scan demonstrated inflammatory changes in the mediastinum with a large amount of soft tissue surrounding the thoracic stent-graft and minimal pulmonary infiltrates (Fig. 1). The first bronchoscopy revealed a small area of granulation in the main left bronchus, but no signs of active or old bleeding. Transbronchial sonography demonstrated a cuff that surrounded the true lumen of the dissected aorta with mixed areas of organized tissue and areas with turbulent blood flow. These findings led us to suspect a communication between bronchial system and the remaining perfused zone of the false lumen of the dissected aorta. At this stage the patient continued to have intermittent bronchial bleeding of approximately 150–250 ml per day.

After an exhaustive evaluation of the clinical, radiological and bronchoscopic findings, and a high suspicion of a connection between the bronchial system and the false lumen dissection, an embolization was deemed necessary.

Through a right common femoral arterial approach in a retrograde fashion, the distal abdominal aorta was cannulated in the true lumen compartment using a superflex 6 F 24 cm long sheath (Optimed, Ettlingen, Germany). No heparin bolus was given intraarterially.

**Fig. 1.** Thoracic CT (August 2003) shows a perfused false lumen of a type B dissection (arrow) and soft tissue surrounding the aortic stent-graft with inflammatory changes in the mediastinum.

A 5F pigtail catheter (Bard Karlsruhe, Germany) was used to carry out an initial angiogram of the infrarenal aorta in order to localize one of the re-entries at this level. Using a 100 cm long 4 F Berenstein catheter (Cordis Miami, USA) and a 0.035″Glide wire (Terumo Tokyo, Japan), we entered the false lumen through one of the re-entries below the renal arteries (3rd lumbar vertebral body) and cranial to the anastomotic segment of the abdominal aortobiliac prosthesis (Fig. 2).

After selective catheterization of the false lumen compartment at the level of the thoracic stent-graft (T5–T10), a new angiogram was performed in this segment. The angiogram revealed multiple areas of abnormal distribution of the contrast material behind the aortic arch in the vicinity of the carina and the main left bronchus and patent intercostal arteries at this level. A very low clearance of the contrast material was observed in the thoracic false lumen segment. No evidence of a direct aorto bronchial fistula was seen (Fig. 3).

Using a FAS-Tracker microcatheter (Boston-Scientific Cork, Ireland) in a coaxial system, the cranial segment of the false lumen was catheterized. Initially, embolization of this segment was performed with six coils ($10\times200\,mm^2$ (3) Boston-Scientific Cork, Ireland), IDC-coils $10\times100\,mm^2$ (2) (Boston-Scientific Cork, Ireland), IDC-coil $9\times100\,mm^2$ (1) (Boston-Scientific Cork, Ireland). After the cranial segment embolization, the microcatheter was positioned more distal in the descending aorta in the vicinity of the tracheal bifurcation, and five more coils were deployed (IDC-coils $10\times100\,mm^2$ (1) (Boston-Scientific Cork, Ireland), IDC-coil $9\times100\,mm^2$ (1) (Boston-Scientific Cork, Ireland) and IDC-coil $9\times200\,mm^2$ (3) (Boston-Scientific Cork, Ireland)).

During this procedure we noted that the coils deployed in the false lumen were moving slowly in cranial direction due to the blood flow with every aortic pulsation, so we decided to bring more stability to this material with another embolic agent. We performed in the same fashion, cranial to distal, embolization with a 1:1 histoacryl (B-Braun Tuttlingen, Germany) and Lipiodol (Guerbet Sulzbach, Germany) mixture using a total of 2 ml histoacryl in the cranial segment and 2 ml histoacryl in the distal segment (Fig. 4).

After a few minutes, we observed stabilization of the embolic material. Immediate angiographic follow-up found significant retention of the contrast material, but there was residual flow in some of the intercostal arteries. We decided to stop the embolization at this moment and wait 2 weeks in order to give the physiological hemostatic mechanisms the opportunity to continue the false lumen thrombosis (Fig. 5).

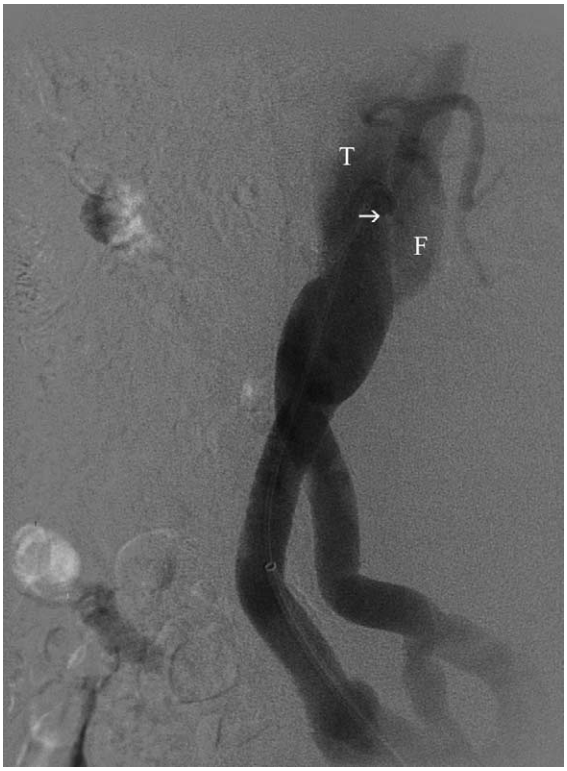


**Fig. 2.** DSA at the abdominal level shows the surgical aortic graft and the entry point of the catheter (arrow) into the false lumen of the dissection (F); (T = true lumen). *HOEHE anastomose und Dissektion gleich*.

**Fig. 4.** DSA shows perfusion of two intercostal arteries and some segments of the false lumen immediately after the embolization. Coils (black arrow) and histoacryl (white arrow) are clearly visible.

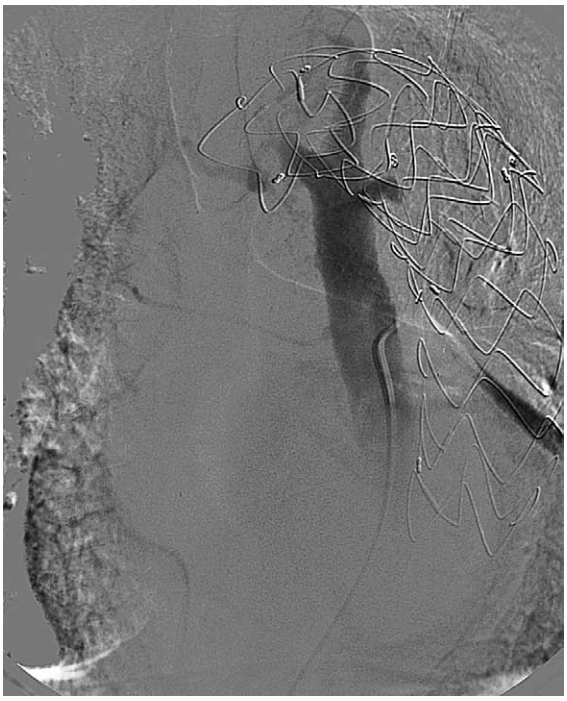

**Fig. 3.** Pre-embolization DSA shows the extension of the false lumen of the dissection behind the aortic stent-graft.

**Fig. 5.** To the right of the aortic arch there is embolization material in the false lumen of the dissection.

The first aortic CTA of the thoracic aorta 26 days after embolization showed complete thrombosis of the embolized false lumen and no evidence of an aorto bronchial fistula or signs of residual pulmonary bleeding. Only a polypoid lesion was observed in the left main bronchus that corresponded to the organized thrombotic material of the last bleeding episode (Fig. 6). This finding was corroborated by bronchoscopy and extraction of the polypoid lesion. No malignancy was found on the pathological examination of the biopsy (Fig. 7).

The patient remained in the surgical care-unit for observation for 4 days and was maintained with the same antibiotic and antihypertensive therapy. No antiplatelet or antihemostatic medication was given. Antibiotic therapy was maintained after the patient was discharged. During his recovery, the patient progressively improved. Clinically his hemoptysis episodes stopped, his fever completely resolved, and his laboratory parameters were improving (red blood cell of 3.6 Pl, hemoglobin 10.9 g/dl, platelets 372/nl, leukocytes 7.26/nl, a PTT 23.3, INR 1.1). The CT scan 1 year after the initial diagnosis showed no perfusion of the false lumen (Fig. 7). The patient remained asymptomatic with no recurrent bleeding.

## Discussion

The association of aortic dissection type A or B and aorto bronchial fistula has been reported in a few cases, especially in those with complicated thoracic aortic pseudoaneurysms and compression of the bronchial system.[1–5] There are no current reports in

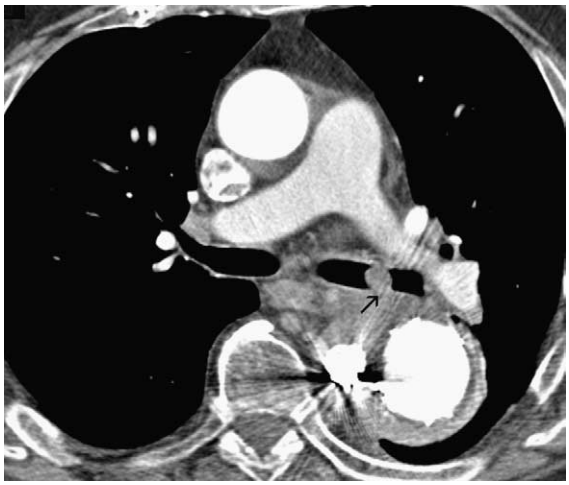

**Fig. 6.** Postembolization CT scan shows embolization material in the false lumen of the dissection. Also there is a polypoid lesion (black arrow) in the main left bronchus. The histopathology was compatible with organized thrombus.

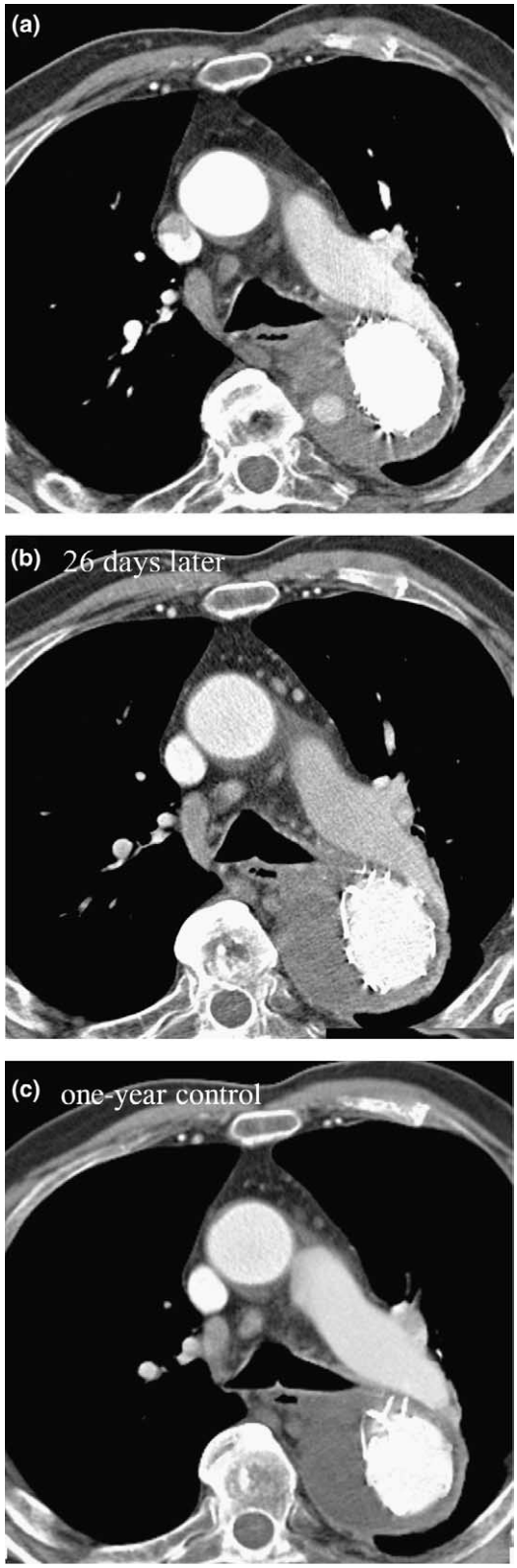


**Fig. 7.** Aortic-thoracic CTA at the same level before (a) and after embolization procedure (b) show thrombosis of the previous perfused false lumen 26 days after the intervention (c) one year CT after initial diagnosis shows permanent thrombosis of the false lumen after the endoluminal embolization.

the medical literature of embolization of the aortic false lumen as a therapeutic option after thoracic aorta stent-graft implantation to close the source of bleeding in progressive hemoptysis. After endoprostheses implantation, the physiologic hemostatic mechanisms tend to thrombose the false lumen of a dissection and progressively reduces false lumen size and pseudoaneurysm rupture risk. However, there is a high variability in the results of endoprosthesis implantation for acute or chronic aortic dissection.[7] Some series report a complete thrombosis of the false lumen in 100% of cases while others report only a 50% success rate 3–6 months after the intervention.[7–12]

Aorto bronchial fistula (ABF) is a common etiology of hemoptysis, especially in those patients who have a previous history of aortic repair. When hemoptysis is massive, mortality is as high as 85%.[13] There are several other causes of hemoptysis including bronchiectasis, thoracic aortic aneurysm, bronchial arterial malformations,[13] mycetoma, tuberculosis, malignancies,[6] Rasmussen's aneurysm,[13] Rendu-Osler-Weber syndrome,[14] polyarteritis nodosa and postradiotherapy fistulas.[15] However, the cause of hemoptysis is undefined in up to 15% of patients.[6] Other reported diseases complicated with ABF and hemoptysis after surgical repair include: aortic coarctation, Fallot's tetralogy, ductus arteriosous or takayasu arteritis. Hemoptysis also complicates bronchial stent implantation.[2]

Current approaches to the study of a patient with hemoptysis must include a detailed clinical history emphasizing the patient's previous cardiac or aortic surgery. The relationship between aortic graft surgery and the development of aorto bronchial fistula mainly in the anastomotic segments are well known.[2,16,17] The hemoptysis can be intermittent or massive. It can vary from a few months to more than 20 years after surgical repair of cardiac or aortic aneurysm.[18] The left lung is the most frequently affected by FAB due to its vicinity to the descendent aorta;[2] however, the series from Swanson reports hemoptysis occurring more frequently in the right lung, especially in the upper lobe.[6]

Radiological diagnosis of ABF should include chest radiography (lung consolidation, or non-specific findings), CT scan (may detect pseudoaneurysm, periaortic hematoma, consolidation of adjacent lung). Angiography should help in the fistula detection.[17,18] Chest X-ray is abnormal in at least 87% of patients.[6] Bronchoscopy excludes other pulmonary causes of hemoptysis and is a more definitive test to identify a bleeding site within the tracheobronchial tree.[6] When a patient with a previous history of cardiac or aortic surgery has hemoptysis, urgent treatment is recommended if the patient also has signs of lung infiltrates on chest X-ray, lung hemorrhage on CT, or visualization of pseudoaneurysm on CT.[2] However, if none of these aforementioned signs appear or they are intermittent, prompt evaluation and eventual treatment are justified. If the bleeding is life threatening, intrabronchial inflation of a cuff into the bleeding site is indicated with a single-lumen endotracheal tube. The tube is positioned in the healthy main stem bronchus, and prompt surgical or endovascular treatment of the primary etiology of the hemoptysis is performed.[2]

Surgical repair has been recognized as a life-saving method for ABF, however, it is invasive with potential severe complications and frequently contraindicated with patients in poor condition.[16] There are reports of endovascular treatment of ABF with *N*-buthyl cyanocrylate plus lipiodol (usually in a one to one dilution).[16] Since, the bronchial circulation is the major source of hemoptysis, therapeutic embolization of bronchial arteries can be performed to stop the bleeding.[6] However, in our case we decided to embolize the false lumen for two reasons: first, the selective embolization of the bronchial arteries was not possible due to the graft interposition, and the previous thoracic angiogram before the embolization which revealed at least two right intercostal arteries (the thoracic level T5–T10) as well as an abnormal collateral net of vessels over the stented aortic arch. Secondly, during the bronchoscopy, transbronchial sonography demonstrated a cuff that surrounded the true lumen of the dissected aorta with mixed areas of organized tissue and areas with turbulent blood flow. These findings led us to suspect a communication between bronchial system and the remaining perfused zone of the false lumen of the dissected aorta.

Many agents have been used to embolize bronchial arteries (i.e. polyvinyl alcohol, coils, sponge particles). *N*-buthyl cianocrilat (hystoacryl B-Braun Tuttlingen, Germany) has also been used in the treatment of other fistulas, such as bronchopleural or esophageal, as well as aortic endoleaks, angiomas, intracranial venous malformations, portal vein and gastroesophageal varices embolization.[19,20] One of the complications of aortopulmonary or ABF is infection with persistent fever, as in our patient. Bacteremia can lead to a subsequent infection of the graft structures, which are difficult to treat. Sometimes the complete removal of all the prosthetic material is necessary. Fortunately, our patient had a very fast response to the antibiotic therapy with the adequate antibiotics. We recommend initiation of broad spectrum antibiotic therapy at the first fever episode with prompt blood culture in order to avoid a secondary graft infection.

There are only a few reports of aortic dissection

complicated with hemoptysis, a rare and often fatal event.[3,21–23] ABF is a uniformly fatal condition unless treated surgical or endovascular.[4,5,24] We think, after deploying an aortic stent-graft for thoracic type B dissection, a good therapeutic option is the embolization of the false lumen, especially in those cases complicated with aorto bronchial fistula.

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